Neurological examination of the unconscious patient

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All unconscious patients should have neurological examinations to help determine the site and nature of the lesion, to monitor progress, and to determine prognosis. The examination consists of observing the patient and eliciting reflexes. We have provided a scheme for the bedside neurological examination of the unconscious patient that can be easily and quickly executed and is easy to interpret. We have not tried to provide a comprehensive pathophysiology of coma; for a more detailed discussion see Plum and Posner[1].

The Glasgow coma scale should be completed (Table 1) and a coma chart initiated to monitor pupillary reactions, pulse, temperature and blood pressure. Neurological examination is most useful in the well-oxygenated, normotensive, normoglycaemic patient with no sedation, since hypoxia, hypotension, hypoglycaemia and sedating drugs profoundly affect the signs elicited.

GENERAL EXAMINATION

Temperature

Pyrexia can be indicative of either infection or medullary lesions.

Hypothermia is a cause of coma.

Neck movements

Neck stiffness is a manifestation of meningeal irritation due to meningitis or subarachnoid blood, or may occur with raised intracranial pressure and coning. However, in the elderly it may only signify degenerative arthritis.

CRANIAL NERVES

Fundi

Papilloedema is a sign of raised intracranial pressure, but is not always present, especially in the acute situation.

Subhyaloid haemorrhages are typically seen in association with subarachnoid haemorrhage.

Diabetic retinopathy

Hypertensive retinopathy

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Pupil size

Small (pinpoint) pupils can be caused by pontine lesions and opioids.

Semidilated (unresponsive) pupils are indicative of midbrain lesions.

Large (unresponsive) pupils are an early sign of oculomotor nerve compression.

Pupil asymmetry

Large, unreactive, pupil is caused by an ipsilateral oculomotor nerve lesion, but could be a pre-existing Adie's pupil.

Small, slow to dilate, pupil occurs in Horner's syndrome. This is a poorly localizing but good lateralizing sign.

Pupil light reaction

A strong light must be used since pupillary responses may be sluggish in unconscious patients (an ophthalmoscope light is not adequate).

Table 1 **Glasgow coma score**

	Score
Best motor response (M)*	
Obeys command	6
Localizes pain	5
Normal flexion	4
Abnormal flexion	3
Extension	2
Nil	1
Verbal response (V)	
Oriented speech	5
Disoriented speech	4
Words only	3
Sounds only	2
Nil	1
Eye opening (E)	
Spontaneous	4
To command	3
To pain†	2
Nil	1

*To central stimulation (see text) and peripheral stimulation by pressure on the nail-bed

†Usually to peripheral pain to trunk (rubbing sternum) or nail-bed pressure

The light reaction depends on an intact pathway from retina via optic nerve to the midbrain then the Edinger–Westphal nucleus, the oculomotor nerve to the ciliary ganglion and finally the pupillary sphincter muscle. A loss of reaction can be due to a lesion anywhere along this pathway, and on its own is poorly localizing but clearly lateralizing.

EYE POSITION/MOVEMENTS

These are dependent upon the oculomotor (III), trochlear (IV) and abducens (VI) nerves.

In the *primary positions*, lesions involving individual nerves can result in a squint (dysconjugate gaze). Seizure activity can result in symmetrical intermittent conjugate gaze in a direction contralateral to the lesion, whilst destructive frontal lobe lesions can result in conjugate gaze to the side of the lesion. Nystagmus is rarely seen, but nystagmus-like movements can occur in status epilepticus. Roving eye movements are usually an indication of light coma and are a poor localizing sign.

To assess *doll's head eye movements* the head is turned from side to side and up and down. The oculocephalic and vestibulocephalic reflexes normally maintain the eye position despite movement of the head, so that the eyes move in the opposite direction to the head movement. Once the movement of the head is complete, the eyes move back to the primary position. The caloric test is more rigorous, but is rarely needed except as part of the assessment of brainstem death.

Abnormal horizontal doll's head eye movements are indicative of lesions affecting oculomotor nerves (III), abducens nerves (VI) and pons.

Abnormal vertical doll's head movements are indicative of lesions affecting oculomotor nerves (III), trochlear nerves (IV) and midbrain.

Vth and VIIth cranial nerves

Corneal reflexes are useful for determining prognosis (Table 2) as well as being helpful for localization.

Absence of corneal reflex is usually caused by a lesion affecting the trigeminal nerve (V), pons or facial nerve (VII).

Grimace to trigeminal pain is assessed by rubbing vigorously anterior to the ear or on the supraorbital ridge. Elicitation of the grimace can be useful to detect upper-motor-neuron facial weakness.

IXth and Xth cranial nerves

These can be assessed by testing for the gag reflex and presence of cough, which depend upon the pathway from glossopharyngeal (IX) and vagus (X) nerves to medulla and then to vagus nerve (X).

Table 2 **Individual signs that indicate very poor prognosis at various stages after onset of non-traumatic coma (adapted from Ref. 2)**

1 day after coma onset	*3 days after coma onset*	*7 days after coma onset*
Absent pupillary light reflex	Absent pupillary light reflex	Absent pupillary light reflex
Absent corneal reflexes	Absent corneal reflexes	Absent corneal reflexes
	Absent motor responses	Extensor or absent motor responses
		Absence of eye opening
		Absence of spontaneous eye movements
		Absence of oculocephalic responses

RESPIRATION

Rapid breathing can be due to pontine lesions as well as respiratory and metabolic causes.

Cheyne–Stokes breathing can be caused by a central medullary lesion or arise from peripheral cardiovascular and respiratory causes.

Irregular breathing can result from medullary lesions.

LIMBS

Tone/posturing

The posture, and posturing to noxious stimuli, should be noted. The noxious stimulus should be applied in a cranial nerve territory, usually either a painful trigeminal stimulus or stimulation of vagal territory during endotracheal suctioning.

Decorticate posture is the equivalent to a bilateral hemiplegic posture with flexion of the arms and hands and adduction of the shoulder. The legs are extended and the feet are inverted and plantarflexed. This can be caused by lesions affecting the pyramidal tracts from the cortex to the internal capsule.

Decerebrate posture is extension of the arms with internal rotation at the shoulder. The position of the legs is

Table 3 **Signs that can help localize brainstem lesions**

Medulla	*Pons*	*Midbrain*
Absent gag and cough reflex	Small, pinpoint pupils	Semidilated unresponsive pupils
Cheyne–Stokes or irregular breathing	Abnormal horizontal doll's head eye movements	Abnormal vertical doll's head eye movements
	Absent corneal reflex	Decerebrate posturing
	Rapid respiration	

Box 1 **Suggested record of neurological examination**

Temperature, pulse, blood pressure, neck stiffness
Glasgow coma score
M, V, E scores
II
Fundi, pupils (size, symmetry, light reaction)
III, IV, VI
Primary position, doll's head eye movements
V, VII
Corneal reflexes, grimace to pain
IX, X
Gag, cough
Breathing pattern
Limbs
Tone/posture/posturing, tendon reflexes, plantars

identical to the decorticate posture. This localizes the lesion to the midbrain, thalamus and subthalamic nuclei, but these in turn may have been distorted by large hemisphere lesions.

Reflexes

Asymmetry in tone, limb reflexes and plantar responses can help lateralize a lesion; symmetrical changes are rarely helpful. It is noteworthy that reflexes and tone may be decreased after an acute upper-motor-neuron lesion.

CONCLUSION

Neurological examination of the unconscious patient not only helps to localize (Table 3) and lateralize lesions but may also be useful in distinguishing causes. Pyrexia and neck stiffness point to subarachnoid haemorrhage, central nervous system infection or coning. Localizing or lateralizing signs without meningism indicate a focal lesion such as tumour, abscess, infarct or haemorrhage. Neuroimaging may be more accurate, but in the acute situation a brief neurological examination can direct and determine appropriate investigation and therapy. Neurological signs can also be helpful in predicting outcome (*see* Table 2)[2]. It is, however, not only the signs at a single point in time that are of importance, but also how they change in time. Along with the coma chart and Glasgow coma scale, sequential neurological examination of the unconscious patient is essential for monitoring progress and should be documented in systematic fashion (Box 1).

REFERENCES

1 Plum F, Posner JB. *The Diagnosis of Stupor and Coma*. Philadelphia: Davis, 1980

2 Levy DE, Bates D, Caronna JJ, *et al.* Prognosis in non-traumatic coma. *Ann Intern Med* 1981;**94**:293–301